# LETTERS TO THE EDITOR

## Staphylococcus lugdenensis and endocarditis

Further to the recent correspondence regarding *Staphylococcus lugdenensis* we report a further case of endocarditis due to this organism.

A 32 year old man presented with a history of fever, rigors for one week preceded by malaise, and weight loss for one month. He described a transient pain and weakness in the right forearm and hand (ulnar aspect) two days before admission. A toolmaker by profession, with no history of rheumatic fever or drug abuse, his only surgery (dental or otherwise) had been a vasectomy three months earlier.

Examination showed that his temperature was 38°C and blood pressure was 130/40 mmHg. Splinter haemorrhages were present on all fingers of both hands. A collapsing pulse was noted. On ausculation signs consistent with aortic regurgitation were found without obvious outflow obstruction, but no signs of cardiac failure. Clinical examination yielded otherwise normal results. Investigations included a white cell count of 12·5 × $10^9$/l, haemoglobin concentration of 13·8 g/l, platelets 152 × $10^6$/l, erythrocyte sedimentation of 70 mm/hour, and C-reactive protein of 19·6 mg/dl. Serum biochemistry and urine examination yielded normal results. An echocardiogram showed a tricuspid aortic valve with a single vegetation.

Staphylococci were isolated from a total of eight blood culture bottles. The slide coagulase test was positive with human plasma and negative with rabbit plasma. The tube coagulase test was negative with both plasmas.

Both Staphylase (Oxoid) and Staphaurex (Wellcome) tests were positive. The plate DNAse test was negative after overnight incubation but positive at five days. API Staph (API Products Ltd, Basingstoke, Hampshire) identified the organism as *Staphylococcus hominis* biotype 1. A positive ornithine test identified it as *Staphylococcus lugdenensis*.[2] The organism was fully sensitive to penicillin, methicillin, aminoglycosides and vancomycin. It was non-phage typeable, using the standard set of phages.

The patient started intravenous flucloxacin (2 g every four hours) plus gentamicin 120 mg twice daily, but within five days he developed early signs of cardiac failure, necessitating urgent valve replacement. At operation a grossly damaged and perforated aortic valve was replaced with a St Jude bileaflet mechanical prosthesis. Postoperative cidal concentrations were inadequate and the patient was changed to benzylpenicillin 1·2 g every four hours plus gentamicin 120 mg twice daily. After three weeks on this regimen he developed a severe allergic reaction to penicillin requiring a change to vancomycin 1 g twice daily plus netilmicin 80 mg twice daily for a further week. The patient made a steady recovery and remains well to date.

Coagulase negative staphylococci cause 5% of native valve endocarditis[3] and of these 28% are on previously normal valves.[4] At 32 years of age this is the youngest reported case of *Staphylococcus lugdenensis* native valve endocarditis.[5] It followed the same aggressive course as those described.[1 5] The transient weakness in the patient's arm suggests an embolic pnenomenon not previously reported in this condition and uncommon in other coagulase negative staphylococci endocarditis.[4] The source of the organism remains unknown, although it is interesting to speculate that it may relate to his vasectomy. Further research is needed to establish the skin distribution and pathogenesis of endocarditis due to this organism.

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1 Smyth EG, Wright ED, Marples RP. New type of staphylococcal endocarditis. *J Clin Pathol* 1988;41:809–14.
2 Freney J, Brun Y, Bes M, *et al.* Staphylococcus lugdenensis sp. nov. and Staphylococcus schleiferi sp. nov., two species from human clinical specimens. *Int J System Bacteriol* 1988;38;168–72.
3 Pelletier LL, Petendorf RG. Infective endocarditis. A review of 125 cases from the University of Washington Hospitals 1963–72. *Medicine* 1977;56:287–313.
4 Geraci JE, Hanson K, Giuliani ER. Endocarditis caused by coagulase negative staphylococci. *Mayo Clin Proc* 1968;43:420–34.
5 Etienne J, Pangon B, Leport C, *et al.* Staphylococcus lugdenensis endocarditis. *Lancet* 1989;i:390.

## Lack of in vitro activity of omeprazole against Campylobacter pylori

There is considerable evidence linking *Campylobacter pylori* infection with gastritis, peptic ulceration, and ulcer relapse.[1 2] We investigated the presence of *C pylori* in 15 patients undergoing diagnostic upper gastrointestinal endoscopy and tested the antibacterial activity of omeprazole, a new anti-ulcer agent,[3] against cultures of *C pylori* obtained from these patients.

Cytology brushings were used to obtain the Gram negative, urease and catalase positive, highly motile bacteria which were cultured microaerophilically (7–8 days at 37°C) for up to five generations on Wilkins-Chalgren agar containing defibrinated horse blood (70 ml/l), amphotericin B (0·02 g/l), cycloheximide (0·5 g/l), trimethoprim (0·25 g/l), vancomycin (0·06 g/l) and nalidixic acid (1 g/l). Electron microscopic examination confirmed that the bacteria were *C pylori* (figure), and was performed on bacteria fixed for one hour in glutaraldehyde (2% in phosphate buffer, 0·1M, pH 7·4), then washed for 30 minutes in the buffer, resuspended in ammonium molybdate (2% in distilled water), and then applied to formvar films coated with carbon and wetted with bacitracin.

*Growth inhibitory activity of omeprazole and furazolidone against four isolates of C pylori*

| Drug concentration (M) | Width of the growth inhibitory zones (cm) Mean (SE) | |
|---|---|---|
| | Omeprazole | Furazolidone |
| $10^{-4}$ | 0·0 | 0·0 |
| $10^{-5}$ | 0·0 | 0·05 (0·05) |
| $10^{-6}$ | 0·0 | 0·25 (0·15) |
| $10^{-7}$ | 0·0 | 2·85 (0·50) |

*C pylori* were obtained, by culture, from five out of six gastric ulcer or gastritis samples, all four duodenal ulcer or duodenitis samples, and five out of eight oesophagitis samples. These findings correlated well with the histopathological inflammatory changes, assessed by haematoxylin and eosin staining (six out of six gastric ulcer or gastritis, two out of three duodenal ulcer or duodenitis, and seven out of seven oesophagitis samples), detected in biopsy samples taken at an adjacent site to the cytology brushings. Detection of the bacterium in the biopsy specimens using Giemsa staining, however, was much poorer (three out of six, none out of three, and none out of seven of the respectively grouped complaints). Twelve out of 15 patients were *C pylori* positive by the culture method; only three out of 13 (two not assessed) were positive by the Giemsa method. Histopathological changes were present in 13 out of 14 patients (one not assessed).

The antibacterial activity of omeprazole (synthesised by Fisons plc) was compared with that of furazolidone (Norwich Eaton Pharmaceuticals, New York, USA), a known inhibitor of the bacterium,[4] against four isolates of *C pylori* using a surface innoculated agar-well technique. Whereas furazolidone inhibited growth of the bacterium, neither omeprazole nor the vehicle control (4% polyethylene glycol 400 in physiological saline) had any effect (table).

Like others,[1 2 5] we found that the organism was present in a high proportion of patients with peptic ulcers or gastritis or duodenitis, as well as in those with reflux oesophagitis.

The superiority of microbiological culture in our hands compared with Giemsa staining for identifying the bacterium may have been related to the sampling techniques used. Given the patchy distribution of *C pylori*, it is more likely that the bacterium will be found by cytology brushing than by biopsy.

If *C pylori* is responsible for ulcer relapse[2] then it seems unlikely on this score that